

UNITED STATES DISTRICT COURT
SOUTHERN DISTRICT OF OHIO
EASTERN DIVISION

IN RE: E. I. DU PONT DE
NEMOURS AND COMPANY C-8
PERSONAL INJURY LITIGATION,

Civil Action 2:13-md-2433
JUDGE EDMUND A. SARGUS, JR.
Magistrate Judge Elizabeth Preston Deavers

This document relates to: *Travis Abbott and Julie Abbott v. E. I. du Pont de Nemours and Company*, Case No. 2:17-cv-998.

EVIDENTIARY MOTIONS ORDER NO. 31

Defendant's Motion to Exclude Testimony of Plaintiff's Specific Causation Expert

This matter is before the Court on Defendant's Motion to Exclude the Specific Causation Testimony of Dr. Kamal Pohar (ECF No. 63, *Abbott* Docket¹), Plaintiffs' Memorandum in Opposition (ECF No. 72), and Defendant's Reply (ECF No. 91). For the reasons that follow, the Court **DENIES** Defendant's Motion.

I.

The litigation between the parties in this multidistrict litigation ("MDL") began in 2001 in a class action in West Virginia state court captioned *Leach v. E. I. du Pont de Nemours & Co.*, No. 01-C-698 (Wood County W. Va. Cir. Ct.) ("*Leach* Case"). The *Leach* Case ended in November 2004 when the parties entered into a class-wide settlement ("*Leach* Settlement Agreement"). (ECF No. 820-8.) In the *Leach* Settlement Agreement, the parties fashioned a unique procedure to determine whether the approximately 80,000 members of the class ("*Leach* Class") would be permitted to file actions against Defendant E. I. du Pont de Nemours and

¹ Unless otherwise noted the ECF references are to the docket in *Abbott*, 2:17-cv-998.

Company (“DuPont”) based on any of the human diseases they believed had been caused by their exposure to ammonium perfluorooctanoate (“C-8” or “PFOA”) discharged from DuPont’s Washington Works plant. The procedure required DuPont and the plaintiffs to jointly select three completely independent, mutually-agreeable, appropriately credentialed epidemiologists (“Science Panel”) to study human disease among the *Leach* Class.

The Science Panel engaged in what ultimately became one of the largest epidemiological studies ever convened, utilizing nearly 70,000 blood samples and medical histories of the *Leach* Class members, and lasting seven years. In 2012, the Science Panel delivered Probable Link Findings for six human diseases (“Linked Diseases”): kidney cancer, testicular cancer, thyroid disease, ulcerative colitis, diagnosed high cholesterol (hypercholesterolemia), and pregnancy-induced hypertension and preeclampsia. The Probable Link Finding means that for the *Leach* Class members it is more likely than not that there is a link between their exposure to C-8 (*i.e.*, drinking water containing at least .05 ppb of C-8 for at least one year) and their Linked Disease. Ultimately, over 3,500 individuals filed cases in this MDL, all of whom alleged that they are *Leach* Class members, are subject to the *Leach* Settlement Agreement, have Linked Diseases, and that C-8 specifically caused their Linked Diseases.

The Science Panel also delivered No Probable Link Findings for approximately 50 diseases it studied. Any *Leach* Class member who received a No Probable Link Finding was prohibited from filing a personal injury action against DuPont as a result of being subject to the *Leach* Settlement Agreement, regardless of whether any other study or expert disagreed with the Science Panel’s No Probable Link Finding.

Beginning in February 2015, this Court held four month-long trials in this MDL: *Carla Marie Bartlett v. E. I. du Pont de Nemours and Company*, Case No. 2:13-cv-170; *David*

Freeman v. E. I. du Pont de Nemours and Co., 2:13-cv-1103; *Kenneth Vigneron, Sr. v. E. I. du Pont de Nemours Company*, Case No. 13-cv-136, and; *Larry Ogle Moody v. E. I. du Pont de Nemours Company*, Case No. 15-cv-803. The first two trials were bellwether trials² and the second two were non-bellwether trials. The parties reached a global settlement of the 3,500-plus cases in February 2017.

Since the global settlement, over 50 cases have been filed (“Post-Settlement Cases”). As did the plaintiffs in the pre-settlement cases, the plaintiffs in these Post-Settlement Cases allege that they are *Leach* Class members, are subject to the *Leach* Settlement Agreement, have a Linked Disease, and that C-8 specifically caused their Linked Disease. Pursuant to the Court’s trial schedule, the parties have filed their motions directed at experts.

II.

A. Expert Testimony

The Federal Rules of Evidence, in particular Rule 702 and 104(a), govern the admission of expert witness testimony and require that the trial judge “ensure that any and all scientific testimony or evidence admitted is not only relevant, but reliable.” *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 589 (1993). Because Rule 702 “requires that the evidence or testimony ‘assist the trier of fact to understand the evidence,’” expert testimony “which does not relate to any issue in the case is not relevant and ergo, nonhelpful.” *Daubert*, 509 U.S. at 590–90. “In other words, there must be a ‘fit’ between the proposed testimony and the question(s) presented by the case at bar.” *Daubert*, 509 U.S. at 591.

² DuPont settled three of the bellwether cases, including *John M. Wolf v. E.I. du Pont de Nemours and Company*, Case No. 2:14-cv-095. (*Wolf* ECF No. 43.) The Court herein refers to some of the briefing from the *Wolf* case.

To determine whether expert testimony is “reliable,” the court’s role, and the offering party’s responsibility, “is to make certain that an expert . . . employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field.”

Kumho Tire Co. v. Carmichael, 526 U.S. 137, 152 (1999). Generally, the expert’s opinions must reflect “scientific knowledge . . . derived by the scientific method,” representing “good science.” *Daubert*, 509 U.S. at 590, 593. The test of reliability is, however, a “flexible” one. *Kumho Tire Co.*, 526 U.S. at 140.

The Supreme Court mandates that a district court exercise its responsibility in acting as the “gatekeeper” for expert testimony. *Daubert*, 509 U.S. at 588; *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 141 (1999). This role, however, is not intended to supplant the adversary system or the role of the jury. *In re Scrap Metal Antitrust Litig.*, 527 F.3d 517, 531–32 (6th Cir. 2008). Arguments regarding the weight to be given any testimony or opinions of an expert witness are properly left to the jury. *Id.* “Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence.” *Id.* (quoting *Daubert*, 509 U.S. at 596).

The burden is on the party proffering the expert report to demonstrate by a preponderance of proof that the opinions of their experts are admissible. *Nelson v. Tenn. Gas Pipeline Co.*, 243 F.3d 244, 251 (6th Cir. 2001). Any doubts regarding the admissibility of an expert’s testimony should be resolved in favor of admissibility. Fed. R. Evid. 702 Advisory Committee’s Notes (“[A] review of the case law. . . shows that rejection of the expert testimony is the exception rather than the rule.”); *Jahn v. Equine Services, PSC*, 233 F.3d 382, 388 (6th Cir. 2000) (stating

that in *Daubert* “[t]he Court explained that Rule 702 displays a liberal thrust with the general approach of relaxing the traditional barriers to opinion testimony”) (internal quotations omitted).

B. Specific Causation Testimony

Defendant moves to exclude the testimony and opinion of Plaintiff’s specific causation expert. As did all of the specific causation experts in the cases in this MDL, Plaintiff’s expert utilized differential diagnosis to reach his ultimate conclusion, which the Sixth Circuit describes as follows:

This circuit has recognized differential diagnosis as an “appropriate method for making a determination of causation for an individual instance of disease.” *Hardyman v. Norfolk & W. Ry. Co.*, 243 F.3d 255, 260 (6th Cir. 2001); *see also Best*, 563 F.3d at 178 (stating that a causation opinion based upon a reliable differential diagnosis may satisfy the requirements of Rule 702). Differential diagnosis is “a standard scientific technique of identifying the cause of a medical problem by eliminating the likely causes until the most probable one is isolated.” *Hardyman*, 243 F.3d at 260 (internal quotation marks omitted). As we explained in *Best*, a physician who applies differential diagnosis to determine causation “considers all relevant potential causes of the symptoms and then eliminates alternative causes based on a physical examination, clinical tests, and a thorough case history.” 563 F.3d at 178 (internal quotation marks omitted).

Pluck v. BP Oil Pipeline Co., 640 F.3d 671, 678 (6th Cir. 2011).

Calling something a ‘differential diagnosis’ or ‘differential etiology’ does not by itself answer the reliability question but prompts three more:

- (1) Did the expert make an accurate diagnosis of the nature of the disease?
- (2) Did the expert reliably rule in the possible causes of it?
- (3) Did the expert reliably rule out the rejected causes? If the court answers “no” to any of these questions, the court must exclude the ultimate conclusion reached.

Id. (quoting *Tamraz v. Lincoln Elec. Co.*, 620 F.3d 665, 674 (6th Cir. 2010)).

“‘The core of differential diagnosis is a requirement that experts at least consider alternative causes.’” *Best v. Lowe’s Home Centers, Inc.*, 563 F.3d 171, 179 (6th Cir. 2009) (quoting *In re Paoli Railroad Yard PCB Lit.*, 35 F.3d 717, 759 (3d Cir. 1994)). Yet, “doctors need not rule out every conceivable cause in order for their differential-diagnosis-based opinions

to be admissible.” *Id.* at 181. “‘The fact that several possible causes might remain uneliminated . . . only goes to the accuracy of the conclusion, not to the soundness of the methodology.’”

Jahn, 233 F.3d at 390 (quoting *Ambrosini v. Labarraque*, 101 F.3d 129, 140 (D.C. Cir. 1996)).

III.

Plaintiff Travis Abbott offers Kamal S. Pohar, M.D., FRCSC as his specific causation expert. DuPont contends that Dr. Pohar’s Expert Opinion and testimony should be excluded because he (1) begins with an assumption or presumption of specific causation, failing to undertake any independent scientific analysis to weigh the respective risks to Mr. Abbot based on his exposure to C-8, (2) bases his opinion on a fiction that the Science Panel made a scientific determination, (3) failed to engage in a reliable differential diagnosis, and (4) did not follow the professional practices that he uses outside the courtroom.

1. An Assumption or Presumption of Specific Causation.

DuPont argues that Dr. Pohar’s expert opinion and testimony should be excluded because he begins with an assumption or presumption of specific causation, failing to undertake any independent scientific analysis to weigh the respective risks to Mr. Abbot based on his exposure to C-8. This is the same argument DuPont made with regard to the plaintiffs’ specific causation experts (Dr. Bahnson, Dr. Gross, Dr. Hanauer, and Dr. Margulis) in the *Bartlett*, *Wolf*, and *Swartz* cases. For example, DuPont argued that “[l]ike Dr. Bahnson, Dr. Margulis started from an *assumption of specific causation*.” (Def’s Mot. to Exclude Bartlett and Wolf Specific Causation Experts: Dr. Bahnson, Dr. Margulis, and Dr. Gross at 26, MDL ECF No. 2823) (emphasis in original). DuPont argued that because the specific causation expert started with the assumption of specific causation, he “did not undertake any independent scientific analysis to weigh the respective risks to Bartlett of her exposure to PFOA and the amount of her increased

risk from other risk factors before ‘ruling out’ the other risk factors, including her morbid obesity.” *Id.* at 26–27. This Court disagreed and explained its decision in detail in EMO 1 and incorporates that explanation here. (EMO 1, Pls’ and Def’s Mots. to Exclude Expert Opinions Related to Causation at 1–3, 6–14, 17–29, MDL ECF No. 4079.)

In the case at bar, Dr. Pohar utilizes the exact same methodology in evaluating Mr. Abbott as did the specific causation experts in *Bartlett*, *Wolf*, and *Swartz*. Dr. Pohar rules in a number of epidemiologically supportable risk and/or causal factors as potential causes of Mr. Abbott’s testicular cancer. He then rules out all potential causes, providing a reasonable basis for their exclusion.

DuPont expands its arguments from those it made in *Bartlett* and *Wolf*, contending:

Dr. Pohar did not determine, and has no opinion regarding, Mr. Abbott’s increased risk for testicular cancer as a result of his exposure to C-8. Pohar Depo. at 94:18-95:9; 103:25-104:6. Dr. Pohar ignores that a *specific* causation expert cannot simply rely on a finding of *general* causation (nor an agreement not to contest general causation) to establish *specific* causation in an individual. Indeed, courts exclude specific causation experts when their opinions “rest primarily” on methodologies “which relate to general causation.” *Lipitor (Atorvastatin Calcium) Mktg. v. Pfizer, Inc.*, 892 F.3d 624, 643 (4th Cir. 2018); *see also Milward v. Acuity Specialty Prods. Grp.*, 969 F. Supp. 2d 101, 108-09 (D. Mass. 2013). As one court has recognized, “[a]n expert . . . cannot merely conclude that all risk factors for a disease are substantial contributing factors in its development. ‘The fact that exposure to [a substance] may be a risk factor for [a disease] does not make it an actual cause simply because [the disease] developed.’” *Guinn v. AstraZeneca Pharms. LP*, 602 F.3d 1245, 1255 (11th Cir. 2010). Assuming a conclusion—as Dr. Pohar did here—is not a reliable methodology for assessing specific causation. For this reason alone, Dr. Pohar should be precluded from testifying on specific causation at trial.

(Def’s Mot. to Exclude Specific Causation Expert Op. and Testimony of Dr. Pohar at 7–8, ECF No. 63.)

This is the same additional argument DuPont made in *Swartz* and this Court addressed in EMO 27, stating:

The case upon which DuPont relies is inapposite. That is, the expert in *Guinn* admitted that the “other risk factors alone were sufficient to explain the onset of [the plaintiff’s] diabetes.” *Guinn*, 602 F.3d at 1249–50. In the instant action, contrary to the expert in *Guinn*, Dr. Margulis does not find that there are other risk factors alone that were sufficient to cause Mrs. Swartz’s cancer.

Further, Dr. Margulis does not simply rely on a finding of general causation, or merely conclude that all risk factors for kidney cancer are substantial contributing factors in its development. Indeed, he did the opposite. Dr. Margulis’ Expert Report reflects his review of numerous scientific studies and other documents, Mrs. Swartz’s medical records, his own physical examination of Mrs. Swartz, which included taking a medical and familial history, and his knowledge, training, and experience as a urologic surgical oncologist. Dr. Margulis utilized differential diagnosis, appropriately “eliminating the likely causes until the most probable one [wa]s isolated,” *Pluck*, 640 F.3d at 678, resulting in his conclusion:

It is my opinion to a reasonable degree of medical certainty, based on my education, training, and experience, together with my review of the documents and information referenced herein, along with my examination of Ms. Swartz, that it is more likely than not that her exposure to C8 was a substantial contributing factor in causing Mrs. Swartz’s RCC [renal cell carcinoma].

(Margulis Expert Report at 8, Swartz ECF No. 27-1.)

(EMO 27, Def’s Mot. to Exclude Pls’ Specific Causation Expert at 9–10, MDL ECF No. 5294.)

The same can be said of Dr. Pohar. That is, the cases relied upon are inapposite and Dr. Pohar does not simply rely on a finding of general causation, or merely conclude that all risk factors for testicular cancer are substantial contributing factors in its development. Indeed, he did the opposite. Dr. Pohar’s Expert Report reflects his review of numerous scientific studies and other documents, his “education, training, and experience” as a urologic oncologist at the James Cancer Hospital and Professor of Urology at The Ohio State University, and his own “independent medical exam, history and physical of Mr. Abbott.” (Pohar Expert Report at 2, ECF No. 64-4.) Dr. Pohar utilized differential diagnosis, appropriately “eliminating the likely causes until the most probable one [wa]s isolated,” *Pluck*, 640 F.3d at 678, resulting in his conclusion:

It is my opinion to a reasonable degree of medical and scientific certainty, based on my education, training, and experience, together with my review of the documents and information referenced herein, along with my independent medical examination of Mr. Abbott, that it is more likely than not that Mr. Abbott's exposure to C8 was a substantial contributing factor in causing both his testicular cancers.

Id. at 9.

Accordingly, DuPont's first argument provides no support for the Court to exclude the specific causation report and testimony of Dr. Pohar.

2. Scientific Determination

DuPont's next argument in support of its request to exclude Dr. Pohar's specific causation opinion is that his "opinions are unreliable because they are based on the false assumption that the science panel made a determination that C-8 was a scientific cause of testicular cancer." (Def's Mot. to Exclude Specific Causation Expert Op. and Testimony of Dr. Pohar at 8, ECF No. 63.) This is the same argument DuPont recently made in its request to exclude the specific causation expert in *Swartz*, which this Court addressed in EMO 27:

DuPont made this same argument related to the Science Panel in its "Overview on Causation" (MDL ECF No. 2813) that it related to all cases in this MDL and in the *Bartlett* case (MDL ECF Nos. 2823, 3203). In DMO 1, DMO 1-A, and EMO 1 this Court found DuPont's argument not well taken, addressing it in detail. (EMO 1, Pls' and Def's Mots. to Exclude Expert Opinions Related to Causation at 6, 9, MDL ECF No. 4079) (DMO 1, Class Membership and Causation, ECF No. 1679); (DMO 1-A, DuPont's Mot. for Clarification on DMO 1 at 11– 14, ECF No. 3972).

As explained in the Reference Manual on Scientific Evidence,³ which DuPont has cited extensively throughout this MDL: "Epidemiology focuses on the

³ "The Reference Manual on Scientific Evidence, here in its third edition, is formulated to provide the tools for judges to manage cases involving complex scientific and technical evidence." *Id.* at xv. "The search is not a search for scientific precision. We cannot hope to investigate all the subtleties that characterize good scientific work. A judge is not a scientist, and a courtroom is not a scientific laboratory. . . The law must seek decisions that fall within the boundaries of scientifically sound knowledge. Even this more modest objective is sometimes difficult to achieve in practice. . . . Judges typically are generalists, dealing with cases that can

question of general causation (i.e., is the agent capable of causing disease?) rather than that of specific causation (i.e., did it cause disease in a particular individual?). *Reference Manual on Scientific Evidence*, Third Edition, Michael D. Green, D. Michal Freedman, and Leon Gordis, Reference Guide on Epidemiology at 552. “Epidemiology has its limits at the point where an inference is made that the relationship between an agent and a disease is causal (general causation) and where the magnitude of excess risk attributed to the agent has been determined[.]” *Id.* at 609 (parenthetical in original). Here, the Science Panel was directed to determine the whether it was “more likely than not” (magnitude of risk) that there was a relationship between C-8 (an agent) and a disease, which resulted in the Probable Link Findings. The study produced by the Science Panel, and funded by DuPont, was one of the largest epidemiological study ever conducted. The physicians were selected by class counsel and DuPont. Without question, Dr. Margulis did not err in relying on the study, the only of its sort, in ruling in C-8 as a potential causal factor in a differential diagnosis. Such an approach did not conflate general and specific causation.

Specific causation, the issue to which Dr. Margulis’s analysis is directed, “is a necessary legal element in a toxic substance case.” *Id.* at 609 (addressing the section on “What Role Does Epidemiology Play in Proving Specific Causation?”).

The plaintiff must establish not only that the defendant’s agent is capable of causing disease, but also that it did cause the plaintiff’s disease. Thus, numerous cases have confronted the legal question of what is acceptable proof of specific causation and the role that epidemiologic evidence plays in answering that question. This question is not a question that is addressed by epidemiology. Rather, it is a legal question with which numerous courts have grappled.

Id. at 609–10 (citing Restatement (Third) of Torts: Liability for Physical and Emotional Harm § 28 cmt. c(3) (2010) (“Scientists who conduct group studies do not examine specific causation in their research. No scientific methodology exists for assessing specific causation for an individual based on group studies. Nevertheless, courts have reasoned from the preponderance-of-the-evidence standard to determine the sufficiency of scientific evidence on specific causation when group-based studies are involved”).

Thus, DuPont’s second argument provides no support for this Court to exclude the specific causation report and testimony of Dr. Margulis.

vary widely in subject matter. Our primary objective is usually process-related: seeing that a decision is reached fairly and in a timely way.

(EMO 27, Def's Mot. to Exclude Pls' Specific Causation Expert Dr. Pohar at 10–12, MDL ECF No. 5294.)

The same analysis holds true here. That is, the Science Panel was directed to determine the whether it was “more likely than not” (magnitude of risk) that there was a relationship between C-8 (an agent) and a disease, which resulted in the Probable Link Findings. The study produced by the Science Panel, and funded by DuPont, was one of the largest epidemiological study ever conducted. The physicians were selected by class counsel and DuPont. Without question, Dr. Pohar did not err in relying on the study, the only of its sort, in ruling in C-8 as a potential causal factor in a differential diagnosis. Such an approach did not conflate general and specific causation.

Specific causation, the issue to which Dr. Pohar's analysis is directed, “is a necessary legal element in a toxic substance case.” *Reference Manual on Scientific Evidence*, Third Edition, Michael D. Green, D. Michal Freedman, and Leon Gordis, Reference Guide on Epidemiology at 609 (addressing the section on “What Role Does Epidemiology Play in Proving Specific Causation?”). As set forth above, and addressed also below, Dr. Pohar's analysis appropriately utilized differential diagnosis to come to his specific causation opinion. Thus, DuPont's second argument provides no support for this Court to exclude the specific causation report and testimony of Dr. Pohar.

3. Differential Diagnosis

DuPont argues that Dr. Pohar failed to “rule in” and improperly “ruled out,” alternative explanations for Plaintiff's testicular cancer. Plaintiff responds

DuPont specifically contends that Dr. Pohar's methodology is flawed because “he failed to rule in or reliably rule out several other risk factors and alternative explanations for Plaintiff's testicular cancer, without any scientifically valid basis for doing so.” (Motion to Exclude at 11). As an initial threshold matter,

it should be noted that this position directly contradicts the sworn testimony of DuPont's own expert.

Q: And again, my question, Dr. Nichols, just to be clear, with his original report and his rebuttal report combined, okay? I'm not asking if you agree with the opinions that he issued. My question is, did [Dr. Pohar] cover all of the risk factors that you believe need to be covered? Did he address them?

A: I think he essentially covered the common risk factors – the commonly listed risk factors, yes.

(Dep. Tr. Of Craig Nichols, MD, June 20, 2019 at 111:18-112:5) (“Nichols Dep. Tr.”) (Aff. Of Jon C. Conlin (“Conlin Aff.”) (filed herewith) Exhibit A).

Q: But again, so my question is, just to be clear, were there – putting aside what your opinion is of his determinations, is there anything he missed in his differential etiology?

A: As I recall, he addressed the issues, the risk factors in one way or another.

(*Id.* at 113:1-6).

(Pls' Mem. in Opp. at 8, ECF No. 72.)

In reply, DuPont argues:

Plaintiffs mischaracterize defendant's expert Dr. Nichols's testimony when they claim that Dr. Nichols “agrees” that Dr. Pohar covered the “commonly listed risk factors.” Plaintiffs' Opp.at 8. Dr. Nichols does *not* agree that Dr. Pohar sufficiently addressed the relevant risk factors. Indeed, Dr. Nichols's testimony critiques Dr. Pohar—in that he “oversimplifies the risk factor setting by only mentioning the older, long recognized risk factors” instead of considering *all potentially relevant risk factors*. See, e.g., Report of Craig R. Nichols, MD, FASCO, FACP (“Nichols Report”) [ECF No. 63] (under seal) at 8. (“The fact that Dr. Pohar did not mention the risk factor of intertubular germ cell neoplasia in the non-ancerous portion of Mr. Abbott's right testicle removed in 2015 *is stunning*”)(emphasis added).

(Def's Reply at 3, ECF No. 91.)

This Court, however, disagrees with DuPont's assessment.

That is, DuPont highlights two risk factors – polygenic and pre-natal roots and

intertubular germ cell neoplasia. As to the first, polygenic and pre-natal roots, DuPont quotes Dr. Nichols' statement that "Dr. Pohar oversimplifies the risk factor setting by only mentioning the older, long recognized risk factors," suggesting that there are newer risk factors that Dr. Pohar missed. However, that is not what Dr. Nichols states in his expert report. Instead, in reviewing Dr. Pohar's report, Dr. Nichols states:

While it may be the case that Mr. Abbott does not have a first-degree male relative with testicular cancer, this hardly reflects the polygenic nature of testicular germ cell cancer. Experts in the field now understand that having a first degree male relative is only a minor component in the story of the heritability of germ cell cancer. The argument that not having an affected first degree male relative rules out a genetic component for an individual affected with testicular cancer is simplistic and false.

(Nichols Exp. Report at 7, ECF No. 64-1.) Dr. Nichols opines:

Dr. Pohar was either unaware or did not feel compelled to mention the tsunami of scientific data now available that strongly implicates polygenic and pre-natal roots for these uncommon diseases and that these concepts are now underpinned by progressively stronger molecular interrogations, epigenetic findings and biological plausibility, and which should be considered as part of any specific causation analysis. . . .

Id. In other words, Dr. Nichols contends that Dr. Pohar did not sufficiently assess the scientific data available for the risk factor polygenic and pre-natal roots – *not* that he failed to address the risk factor as DuPont contends.

Similarly, the other risk factor highlighted by DuPont is intertubular germ cell neoplasia ("cancer in situ" or "IGCN" or "GCNIS"), which DuPont suggests was not even mentioned by Dr. Pohar by quoting Dr. Nichols statement: "The fact that Dr. Pohar did not mention the risk factor of intertubular germ cell neoplasia in the non-cancerous portion of Mr. Abbott's right testicle removed in 2015 is stunning." (Def's Reply at 3, ECF No. 91, and quoted above.) However, Dr. Pohar did address this risk factor as part of his differential diagnosis, stating that the one of the "well established risk factors" is the "presence of intratubular germ cell

neoplasia.” (Pohar Report at 9, ECF No. 64-4.) In rebuttal to Dr. Nichol’s suggestion that he did not put a sufficient amount of weight on intertubular germ cell neoplasia, Dr. Pohar states in part:

Dr. Nichols’ report places tremendous emphasis on prenatal factors and inherited genetic influences as the root cause of the majority of germ cell testicular cancers, including both of Mr. Abbott’s testicular cancers. Dr. Nichols’ report implies that intratubular germ cell neoplasia (IGCN) is the obligate precursor of essentially all germ cell cancers, including both of Mr. Abbott’s testicular cancers, and invariably IGCN arises in the fetal testis and is present before the male child is born. Based on his knowledge of gonad development and descent and hormonal signaling in the fetal gonad, Dr. Nichols incorrectly postulates that IGCN must arise in the context of a testicular dysgenesis syndrome that manifests early in embryogenesis. . . .

I disagree with the logic and interpretations of Dr. Nichols on many salient points related to causation of testicular cancer in reference to Mr. Abbott. As Dr. Nichols states, there have been many recent studies investigating the possibility of a genetic predisposition (i.e. causative germ line mutation) for testicular cancer and none have identified a highpenetrance, single-driver gene alteration in the pathogenesis of germ cell cancers. . . .

The fact that Mr. Abbott had IGCN detected in his orchiectomy specimen in 2015 should be no surprise. In over 90% of postpubertal germ cell cancers this entity is found in the surrounding normal testicular parenchyma, both seminoma and non-seminoma, and is the well known obligate precursor of adult testicular cancers. Its presence in the 2015 radical orchiectomy specimen has very little relevance in determining causation of Mr. Abbott’s testicular cancers

(Pohar Expert Rebuttal Report at 2–4, ECF No. 64-5.)

Thus, DuPont is incorrect that Dr. Pohar failed to address polygenic and pre-natal roots and intertubular germ cell neoplasia. DuPont’s expert merely disagrees with the weight Dr. Pohar accorded these risk factors. Accordingly, DuPont’s third argument provides no support for this Court to exclude the specific causation report and testimony of Dr. Pohar.

4. Professional Practices

DuPont’s final argument for exclusion of Dr. Pohar’s opinion and testimony is that he did not follow the professional practices that he uses outside the courtroom. Specifically, DuPont argues:

Dr. Pohar conceded that, in his regular professional practice of medicine outside the courtroom, he treats patients with testicular cancer but does *not* determine the cause of their cancer and, therefore, does not tell them that one thing was the cause or a substantial contributing cause of their testicular cancer. *See* Pohar Depo. at 194:7-20, 197:13-198:1, 267:1-8 (“Q. . . . As a treater, though, in how many instances of the approximate 300 testicular cancer patients that you’ve seen have you determined the cause of your patient’s testicular cancer? [. . .] A. I mean, I haven’t determined the cause of any patient’s testicular cancer . . . in my – in my medical practice delivering care.”).

(Def’s Mot. to Exclude Specific Causation Expert Op. and Testimony of Dr. Pohar at 13, ECF No. 63.)

DuPont concludes:

For this additional reason—that the made-for-litigation approach he used here differs from the approach he has used in his regular professional practice outside the courtroom—his opinions on specific causation should be excluded. *See, e.g., Simmons v. Novartis Pharms. Corp. (In re Aredia & Zometa Prods. Liab. Litig.)*, 483 F. App’x 182, 190 (6th Cir. 2012) (courts should view “with special caution expert testimony prepared solely for purposes of litigation, rather than flowing from an expert’s line of scientific or technical work”); *United States EEOC v. Rockwell Int’l Corp.*, 60 F. Supp. 2d 791, 797 (N.D. Ill. 1999) (excluding expert testimony where the expert “failed to employ the same level of intellectual rigor that characterizes the practice of experts in his field, or even his own normal practice.”); *In re Silica Prods. Liab. Litig.*, 398 F. Supp. 2d 563, 639 (S.D. Tex. 2005) (excluding expert testimony where “the gulf between” the methodology used in the litigation and the methodology the expert advocated for in his academic work “starkly contravene[d]” the requirements of *Daubert*).

Id. at 13–14. This is the exact argument DuPont made as to the specific causation expert in *Swartz*, which this Court addressed in EMO 27, stating:

DuPont’s argument misses the mark. That is, DuPont does not contend that Dr. Margulis failed to employ the same level of intellectual rigor that characterizes the practice of experts in his field. Instead, DuPont contends that Dr. Margulis’s regular work does not include providing expert opinions, but rather in his professional practice he treats patients. This fact, however, has no impact on the admissibility of Dr. Margulis’s specific causation opinion, which DuPont does not claim he is unqualified to make.

Dr. Margulis was asked to determine whether C-8 was a substantial contributing cause of Mrs. Swartz’s kidney cancer. Dr. Margulis made that determination. The contention that in his role as the Chief of Urology at Parkland

Memorial Hospital he is not asked to perform differential diagnoses to determine whether a particular risk or causal factor was a substantial contributing cause to the cancer for which he is treating a patient does nothing to take away from his qualifications to make such an inquiry. Indeed, DuPont does not argue that Dr. Margulis is unqualified to make such a determination. DuPont's argument merely underscores that, in his medical practice, Dr. Margulis does not typically offer medical opinions on legal issues.

The cases upon which DuPont relies are unhelpful. In *Rockwell International*, the court excluded an expert's opinion because the expert admitted that he "failed to employ the same level of intellectual rigor that characterizes the practice of experts in his field, or even his own normal practice." *U.S. E.E.O.C. v. Rockwell Intern. Corp.*, 60 F. Supp. 2d 791, 797 (N.D. Ill. 1999), *aff'd sub nom. E.E.O.C. v. Rockwell Intern. Corp.*, 243 F.3d 1012 (7th Cir. 2001). The court explained,

It is obvious that Brethauer, who candidly admitted such at the *Daubert* hearing, was directed to employ principles that contradicted his normal methodology in various respects. He performed analyses he would not normally perform. He included analyses that he would not normally include. He included calculations upon which he did not rely and did not fully believe should be followed. He relied on materials, reports and summaries given to him by counsel, and failed to verify the information from reliable, independent sources. Finally, he incorporated language drafted by [Attorney] Waldron. It is one thing for lawyers to make authorized revisions to an expert's prepared report. *See Marek v. Moore*, 171 F.R.D. 298 (D.Kan.1997). It is quite another for an expert to include calculations upon which he did not rely and he would not rely on simply to appease his client's attorney. A proffered expert must "bring to the jury more than the lawyers can offer in argument." *Salas v. Carpenter*, 980 F.2d 299, 305 (5th Cir.1992).

Id.

In the present action, DuPont makes no argument that Dr. Margulis did even one of the actions the Seventh Circuit found objectionable. DuPont does not argue that Dr. Margulis was directed by counsel to employ principles that contradicted his normal methodology in any respect, that he utilized a methodology he would not normally utilize if asked to perform a causation analysis, that he included calculations upon which he did not rely and did not fully believe should be followed, or that he relied upon materials, reports and summaries given to him by counsel that he failed to verify.

Similarly, in *In re Silica Prods. Liability Litigation*, the court explained the requirement that "an expert . . . employ[] in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." 398 F. Supp. 2d at 622 (quoting *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137,

152 (1999)). There is no suggestion here that Dr. Margulis failed to employ the same level of intellectual rigor that characterizes the practice of an expert in the relevant field. Again, DuPont makes no argument that Dr. Margulis is unqualified to engage in the causation analysis he was asked to complete.

Thus, this last argument too provides no support for DuPont's request to exclude the specific causation report and testimony of Dr. Margulis.

(EMO 27 at 16–18, ECF No. .)

The same analysis is applicable here. That is, DuPont does not contend that Dr. Pohar failed to employ the same level of intellectual rigor that characterizes the practice of experts in his field. Instead, DuPont contends that Dr. Pohar's regular work does not include providing expert opinions, but rather in his professional practice he treats patients. This fact, however, has no impact on the admissibility of Dr. Pohar's specific causation opinion, which DuPont does not claim he is unqualified to make. And, likewise, the cases relied upon by DuPont are the same and for the same reasons stated in EMO 27 are unhelpful. Consequently, this last argument also provides no support for DuPont's request to exclude the specific causation report and testimony of Dr. Pohar.

IV.

For the reasons set forth above, the Court **DENIES** Defendant's Motion to Exclude the Specific Causation Testimony of Dr. Kamal Pohar. (Abbott ECF No. 63.)

IT IS SO ORDERED.

1-2-2020
DATE



EDMUND A. SARGUS, JR.
UNITED STATES DISTRICT JUDGE